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RENAL TUBERCULOSIS *

WHY CLINICALLY ESTABLISHED RENAL TUBERCULOSIS NEVER COMPLETELY HEALS

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DISCUSSION *by Edward L. Keyes, M. D., New York City; Arthur L. Chute, M. D., Boston; Alexander Randall, M. D., Philadelphia.*

THIS paper is intended to present an hypothesis based on accepted facts. The word "never," appearing in the title, is not intended to be taken literally but to signify extreme rarity.

SOME OF THE FACTS INVOLVED

Many physicians still resist the significance of the well-known clinical fact that a tuberculous kidney, definitely discharging tubercle bacilli, represents a progressively destructive process, and that there are few, if any, authentic records of complete healing in such a case. They seem to reason that because apparently complete healing does occur not infrequently in other organs, renal tuberculosis should not constitute an exception. If we can make them understand and appreciate the underlying reason for this difference, perhaps there will be less temporizing in cases of unilateral renal tuberculosis at a time when nephrectomy would probably result in a cure.

In deducing the hypothesis which is submitted in this paper, other questions unavoidably creep in, particularly concerning the origin and early developmental phenomena of upper urinary tract tuberculosis. Again, there arises the following pertinent query: Being of hematogenous origin, why is the disease bilateral in only 35 per cent of cases? Are bacilli in sufficient numbers carried by chance to only one kidney? Or do the organisms reach both kidneys, but gain a foothold in one only?

WHEN RENAL TUBERCULOSIS IS CLINICALLY ESTABLISHED

When pus and tubercle bacilli of *renal* origin are found in the urine, renal tuberculosis may be said to be "clinically established."

FACTORS WHICH DELAY HEALING BY CICATRIZATION

In several organs or tissues of the body other than the kidney, recovery or complete healing by cicatrization is a frequent occurrence. Why the difference in the case of the kidney? The answer seems obvious, one reason perhaps why writers have neglected to comment on this phase of the subject.

Renal tuberculosis is always secondary to a tuberculous focus elsewhere in the body, and the infecting organisms almost invariably reach the kidney by way of the renal artery. The lesion originates in the parenchyma. The first gross evidence is usually observed in a pyramid near its base. So far as may be inferred from animal experimentation, microscopic tubercles in the cortex may antedate this gross lesion in a pyramid by a considerable period of time. Until there is an ulcerative lesion, that is to say until the tuberculous process has extended to a calyx or to the collecting tubules, tubercle bacilli are not present in the urine from that kidney, nor does the urine contain pus cells at this stage unless the pus cells arise from a preëxisting or concomitant nontuberculous infection. Finding of acid-fast bacilli without pus cells is not evidence of urinary tuberculosis. In the prepyuric stage there are rarely any pronounced subjective symptoms or objective findings referable to the urinary tract. There may be a slight albuminuria or an afternoon rise of temperature, seldom over 99.2 degrees, unless due to a tuberculous lesion elsewhere or other concomitant cause—rarely anything pronounced until pus cells appear in the urine. Even a slight soreness over the kidney is uncommon in the prepyuric stage. Tenderness and pain over or in a tuberculous kidney means that the tuberculous process is not recent. Indeed, renal tuberculosis has usually existed from one to three years before it is definitely diagnosed. Excluding other etiological factors, frank hematuria of renal origin means that the disease has already invaded or, by reason of a ruptured vessel, has suddenly broken into the renal pelvis.

When a tuberculous process once reaches a calyx, a new clinical picture, as well as characteristic pathologic changes, soon begin, due to a descending infection involving the ureter and bladder. Tubercle bacilli are now being excreted,

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whereas in the prepyuric stage these bacilli were not coming down. The new clinical manifestations, essentially urinary, are frequency and dysuria of greater or less degree, and pyuria, and these are the symptoms for which the patient usually first consults his physician. The condition is all too frequently diagnosed at this time as just "cystitis," without any adequate attempt to identify the infecting organism or determine the primary focus in the urinary tract.

The ureter almost inevitably becomes the site of characteristic pathologic changes, viz., tubercles, infiltration, ulceration, thickening, fibrosis, and rigidity, which lead to constriction of its lumen at one or more sites. Besides the stenosis or stricture, the peristaltic function is interfered with. In some degree, probably, deficient elasticity must be present in even the mildest form of ureteral tuberculosis. As a result of a constricted ureter and defective peristalsis, a marked degree of back-pressure is produced in the kidney. It is scarcely rational to expect the recovery of a kidney handicapped by such hindrance to drainage of its never-ceasing secretion. Ureteral dilatation is of little, if any, lasting therapeutic value, in contradistinction to ureteral stricture produced by a nontuberculous process.

The point is often made that with rest, fixation, heliotherapy, and other measures, tuberculosis heals through the process of sclerosis, calcification, etc. In this respect renal tuberculosis differs from that in practically every other organ where tuberculosis is common, because there can be no rest for either kidney or ureter. This, plus ureteral stenosis, seems to be the answer to the tuberculous kidney's unfailing and ultimate destruction, not inconsistent with the fact that under accepted therapy healing does take place in other organs.

By careful section of tuberculous kidneys there is abundant evidence of healing, healed areas being found side by side with tubercles and all the other pathologic changes incident to tuberculosis. But in spite of these localized attempts at healing, new areas become involved and the tuberculous process in the kidney is always progressive, once the tubercle bacilli reach a calyx. On the other hand, there is some supporting evidence (Chute and others) and much reason to believe that small tuberculous lesions in the renal cortex do sometimes heal completely; but never when the lesion has reached a pyramid or calyx, or collecting tubules. Even the observations of Wildbolz, Medlar, and Thomas and Kinsella, each with his somewhat unorthodox individual theory on certain aspects of the disease, dovetail with this hypothesis when carefully analyzed. Animal experimentation is apt sometimes to lead to erroneous conclusions for the reason that in the case of man, in addition to other possible factors, evolution and civilization have conspired to add another unfavorable influence, namely, the upright position, often producing a sagging kidney and ureteral kink. Sagging and the resultant interference with drainage, predispose to pyogenic infection in general, and to renal tuberculosis in particular, if the blood stream should happen to contain tubercle bacilli. As has been particularly emphasized by Frand Kidd, probably most of us intermittently carry a few organisms in our blood stream, and a kidney handicapped by slight hindrance to drainage and an increase in intrapelvic pressure is apt, by selectivity, to become infected, while its fellow on the opposite side escapes or recovers rapidly. A striking example is unilateral typhoid pyelonephritis. When the blood stream is swarming with *B. typhosus,* why should one kidney escape? In one instance of unilateral typhoid pyelonephritis, the writer was enabled to indicate by means of ureteropyelography the plausible explanation and probable reason why one kidney became infected, and the other escaped: a handicap to drainage on the affected side by reason of a moderate obstruction in its ureter, and hence its especial vulnerability.

By the same token it is logical to suppose that in a large percentage of cases of unilateral renal tuberculosis the bacilli reach both kidneys, but that in the vulnerable kidney the process is progressive while in the other the infection is overcome before it extends to the medulla. We can hardly escape the conclusion that when tubercle bacilli are present in the blood stream in sufficient numbers to infect a kidney, probably they are carried to the opposite kidney as well as to other organs; but that there exists a special reason or factor that enables infecting organisms to gain a foothold in that particular kidney.

SUMMARY

1. Where a tuberculous lesion has advanced to the renal pelvis or collecting tubules, renal tuberculosis never completely heals because of the handicap of hindered drainage and back-pressure, due to tuberculous changes in the ureteral wall; that is to say that, aside from other possible reasons, a tuberculous kidney excreting tubercle bacilli cannot recover because an incurably strictured ureter interferes with normal drainage and peristalsis.

2. When complete healing does occur, the tuberculous process is limited to the cortex, *i. e.,* has never extended to a pyramid or collecting tubules.

3. Until the tuberculous process has extended to a calyx or the collecting tubules, pus and tubercle bacilli of renal origin will not be found in the urine and therefore, up to this time, renal tuberculosis cannot be said to have been "clinically established."

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DISCUSSION

EDWARD L. KEYES, M. D. (Department of Clinical Surgery-Urology, Cornell University Medical College, New York City).—Some of the observed facts concerning the healing of renal tuberculosis are:

1. At tuberculosis sanatoria routine repeated examination of urine from patients with active pulmonary tuberculosis will occasionally reveal tubercle bacilli and, more rarely, tubercle bacilli and pus.

2. When such patients are followed to autopsy the bacilluria cases as a rule show no tuberculous renal

lesion on routine pathological examination. The pyuric cases show tuberculous lesions somewhere along the urinary tract.

3. The urologist occasionally encounters cases of arrested renal tuberculosis in the form of:

(*a*) Complete silent caseation of a kidney whereby its function is totally lost, though if the process has not extended to the ureter and if there is no mixed infection the patient may die without recognizable symptoms or detriment due to this lesion;

(*b*) Partial or temporary healing of active clinical tuberculosis of the kidney; I have reported two cases, one with an entire remission in symptoms for six years, the other a remission for three years, both submitting to nephrectomy for recurrence of active lesions.

(*c*) Unexplained cases fully diagnosed as active surgical tuberculosis whose symptoms cease over a long period of years and who refuse further examination.

(*d*) Pus and tubercle bacilli are obtained by ureter catheter from each kidney. One kidney is removed. The patient is not technically reëxamined. He remains clinically well for a period of more than ten years.

4. All of these occurrences are extremely rare. I have followed over two hundred cases of renal tuberculosis. I have seen three "pathological nephrectomies," one of which had begun to show activity, which required surgical nephrectomy. I have not noted any other clinical remissions as prolonged as those I have reported. I have observed one case of unexplained prolonged remission of tuberculosis in one kidney and one in the remaining kidney after nephrectomy for tuberculosis.

5. Nephrectomy for tuberculosis is an operation with extraordinarily low mortality. This has varied in my experience and that of others from two to five per cent in large series of cases. Except for prolonged healing of the wound which complicates about five per cent of the cases, the convalescence from operation may be expected to be entirely peaceful; much more so, for example, than convalescence from nephrotomy for stone. Refusal of nephrectomy for the cure of unilateral tuberculosis means that the patient is willing to face the prospect of a miserable death from bladder tuberculosis with not more than a two per cent possibility of escape.

6. For twenty years urologists have demanded of the medical men who encourage nonsurgical treatment of renal tuberculosis a justification in the form of a postmortem pathological specimen of surgical renal tuberculosis that showed evidence of complete healing without complete destruction of the kidney. Such a specimen has not been forthcoming.

7. It is for these reasons that the urologist refuses to accept medical treatment in substitution for nephrectomy.

8. Nevertheless it is sometimes profitable to spend a number of weeks in establishing a precise diagnosis of renal tuberculosis and it is not justifiable to perform nephrectomy for bacilluria without pyuria or deformity of the renal pelvis.

9. An interesting theory covering the origin and development of renal tuberculosis is that of Medlar, who made serial sections of the kidneys of patients dying from pulmonary tuberculosis. He found cortical evidences of multiple tuberculous inoculation of both kidneys as mentioned by Doctor Day. These lesions showed either—

(*a*) Regression and healing without caseation (and in some instances this was associated during life with bacilluria); or

(*b*) Caseation, or suppurating because of mixed infection. These lesions extended to and broke into the pelvis, discharging pus and tubercle bacilli.

Class (a) may be termed medical renal tuberculosis. It is characterized by being bilateral and showing rare tubercle bacilli, but no pus in the urine. Class (b) is surgical tuberculosis characterized by pus in the urine at least at times. (One exceptionally sees extensive renal tuberculosis without pus or tubercle bacilli in the urine during periods of inactivity.)

10. Medical tuberculosis is characterized by a normal pyelogram; surgical tuberculosis by an abnormal pyelogram.

11. Surgical tuberculosis at onset is habitually unilateral because:

12. Medical tuberculosis may heal in both kidneys—usually heals in one.

13. Surgical unilateral tuberculosis calls for nephrectomy to save the patient from a miserable death by bladder tuberculosis.

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Arthur L. Chute, M. D. (Department of Urology, Tufts College Medical School, Boston, Mass.)—Doctor Day's paper has been of especial interest to me since it brings up some of the points that I considered in a paper I read before the American Urological Association in New York in 1920. I believe, as does Doctor Day, that the tuberculous infection of a kidney must always be secondary to some other tuberculous lesion of the body, and that the only way the infection can reach the kidney is through the blood stream. Furthermore, we both believe that it is practically impossible for all the tubercle bacilli that are thrown into the blood stream, from the primary focus, to be carried to one kidney, that though both kidneys probably receive a more or less equal number of bacilli, but one is infected in about 65 per cent of the cases, due to the fact that there must be some condition of lowered resistance in one kidney that does not exist in the other. This may be an anatomical variation or some more or less intangible thing that produces a lowered resistance in one organ. I agree with this, but I also think that kidneys have a considerable power to combat a beginning tuberculous infection, especially one that takes place in the cortex, and cited in my paper two cases of patients with vague discomfort in one loin, in each of which a radiograph unfortunately showed a small round shadow which moved with the kidney; in one of these I curetted the small calcified area and stirred up what was evidently a healing tuberculosis, so that a nephrectomy had to be done shortly after and the man was ill for about a year with various tuberculous manifestations; the other patient was similarly treated by another man and led to a fatal dissemination of tuberculosis in a few weeks. I cited these cases as ones in which a beginning tuberculous process was being stamped out until nature's healing process was interfered with. I believe this is what happens more or less often in a beginning renal tuberculosis and we know nothing about the infection.

Doctor Day rightly says that after a tuberculous renal infection has reached the stage where pus and tubercle bacilli are found in the urine, the process is rarely cured in the way that we see cure in other renal suppurations: that is, cicatrization of the lesion with a secreting kidney. He attributes this progression of the process to the influence of back-pressure, due to the thickened ureter, or to a tuberculous stricture of the ureter. When present this obstruction may be a factor, but in a certain number of cases I have removed a badly infected tuberculous kidney where a ureter catheter passed up the ureter without obstruction and when at least the upper part of the ureter was so thin and flexible that I hesitated to cut it. When, and if present. I do not doubt that a thickened and strictured ureter may add to the destructive process. On the other hand, it has been in the instance of ureters that were thickened to the point of being occluded that I have seen so-called autonephrectomy take place; in one instance of this sort the patient, from the history, had gone five to six years without symptoms with a shut off right ureter. As it

became painful after that time I did a nephrectomy, finding a completely shut off kidney. She died eleven years later, supposedly of Addison's disease of the other side. No autopsy was obtained.

I believe the "unfailing and ultimate" destruction of the tuberculous kidney is due to the fact that we can only recognize renal tuberculosis when pus and tubercle bacilli are in the urine and when, therefore, it is really relatively advanced: that the beginning cases are the ones to which I have referred. I do not agree with the first conclusion of the writer's summary that a strictured condition of the ureter is the chief reason for the inexorable progress of a renal tuberculosis once it has advanced to the point where it is recognizable. Besides the above this condition of thickened ureter is not always present in a progressive case. With the writer's second point I agree perfectly, but I should put it in a little different way, namely, that tuberculous infections of the cortical part of a kidney are presumably more common than we suppose and many get well of themselves without being recognized. I quite agree with the third heading of his summary, and hope the time will soon come when we shall have symptoms to guide us to the recognition of the incipient stage of renal tuberculosis. In my belief, though I cannot prove it, many of these patients recover without treatment, and if we could only recognize the condition and give them proper hygienic treatment I think we would greatly diminish the really advanced cases which I believe are the only ones we recognize surgically, and which must, as a rule, be treated by nephrectomy. Doctor Day's paper brings up certain hypotheses that need more observation and study than we have given them.

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Alexander Randall, M.D. (Department of Urology, University of Pennsylvania School of Medicine, Philadelphia, Pa.)—Doctor Day has put upon a logical and clinical basis ideas and facts that experimenters have long tried to impress upon clinicians. In 1917 Noel Hallé (Presse Médicale) presented the first acceptable classification of renal tuberculosis on a biological basis. His pathological classification was almost too simple (as compared to those in current textbooks) for its immediate acceptance. His work, however, has since been repeated and amply substantiated by subsequent students of the problem. Hallé allowed but two types of chronic renal tuberculosis (all agreeing as regards the acute miliary disease). The first is chronic closed parenchymatous renal tuberculosis, and the second, chronic open pyelitic renal tuberculosis. Note, if you will, the careful incorporation of the adjectives "closed" and "open." It is the latter variety (the chronic open pyelitic renal tuberculosis) that Doctor Day identifies as being "clinically established" and he is right: others call it "surgical" renal tuberculosis, while all recognize it as a progressive, destructive, and incurable lesion. Surgery is its only hope and, fortunately, it is predominately unilateral in its early stages. The first variety (chronic closed parenchymatous renal tuberculosis) holds out interest when Hallé states that 33 per cent of these cases heal spontaneously, and says we fail to recognize in the so-called renal infarcts the scars of healed parenchymatous tuberculosis. Doctor Day has shown you how, clinically, in this type the evidences in the urine are scarcely recognizable and perhaps we are right in calling it, for contradistinction, "medical" renal tuberculosis, for until our diagnostic methods advance, its clinical recognition is presumptive only and its treatment expectant and medical. If the profession grasps these hard but simple facts, founded upon competent research and clinical investigation and study, the delay in operating upon an inevitably progressive lesion will be decreased, with a prevention of complications and spread of disease, while the ultimate relief and cure will be proportionately increased.

Doctor Day (Closing).—I am especially privileged in that three such renowned authorities as Doctors Chute, Keyes, and Randall should discuss my small contribution concerning one phase of renal tuberculosis.

Some observations of Doctor Chute's (President's Address, American Urological Association, *Journal of Urology*, May, 1921) were the starting point of this hypothesis, which after all is intended as a simple clinical clarification of some aspects of renal tuberculosis in conformity with Hallé's conception of its pathology.

The reader should clearly recognize the full import of one sentence especially, as stated in my paper, viz.: "When pus and tubercle bacilli of *renal origin* are found in the urine, renal tuberculosis may be said to be 'clinically established.'" The proof that the pus and tubercle bacilli *are* of renal origin is often a highly technical urologic procedure and its comprehensive discussion is out of place here. Suffice it to say that tubercle bacilli, having their source in the genital adnexa, may be present in the urine with absolutely no lesion in the kidney. Even tuberculosis of the lower ureter has been observed in a few instances with no involvement of the kidney whatever, perhaps carried upward from the genital adnexa through the lymphatics.

Doctor Chute states that he disagrees with the postulate that a strictured ureter is the chief reason for the inexorable progress of renal tuberculosis. I shall have to modify the title of my hypothesis slightly to meet his sound objection. He believes that by the time the condition is clinically diagnosed the process is so extensive that recovery is quite impossible. What, subconsciously, I meant to convey, and should have stated in my title is, "Why clinically established renal tuberculosis *cannot* recover." For if it were otherwise possible for any case of renal tuberculosis to heal completely, the tuberculous ureter would constantly threaten any remaining chance for the kidney to recover, even if nature had successfully negotiated all other hurdles. Israel, eminent German urologist and urological pathologist, among numerous other observers, endorses Caspar's statement that "In renal tuberculosis of long standing the ureters invariably become diseased."

In order to make my paper lucid and avoid confusion, I purposely avoided discussing the exceptions. But the exceptions prove the rule. Doctor Keyes brings them up in the mention of remissions and autonephrectomy. In his two cases where long remissions were noted, nephrectomy, however, was necessary in the end, because the kidney had never healel completely. His autonephrectomy cases with clinical cure comprised one per cent of the total, perhaps higher than the average. Crenshaw, in a review of 1,817 cases observed at the Mayo Clinic, reports only a few cases of total calcification and autonephrectomy which were not nephrectomized and over an average period of thirty months were classed as "practically well" or "improved." These cases comprise only four-tenths per cent of the total number. Neither remissions nor autonephrectomies constitute cures, that is to say, complete healing. In autonephrectomy cases the incidence of metastases through the blood stream, such as acute miliary tuberculosis, tuberculous meningitis, etc., is itself proof of the fallacy of the doctrine of healed lesions in such instances, as stressed by Wildbolz.

Tubercle bacilluria with pus, of which Doctor Keyes speaks, has been reported in sanitoria cases as he says. The overwhelming consensus of opinion is that where the bacilli are of renal origin there is an ulcerative tuberculous lesion in the kidney which was overlooked at autopsy. Medlar is very emphatic on this point. Medlar's careful serial sectioning, which for persistency, thoroughness, and conscientiousness is unexcelled, would have probably demonstrated tuberculosis in such kidneys, and in those which Wildbolz classifies as "tuberculous nephritis." In series of over 1,000 observed cases, 660 of which he nephrectomized,

Wildbolz was able to report, however, only four cases which he himself classed as tuberculous nephritis that had gone over a five-year period—less than one-quarter per cent. His criteria for diagnosis are sparse tubercle bacilli demonstrated by guinea-pig inoculation but undiscoverable in stained smears from the urine. A further proviso is that the urine is free of pus cells. The slight incidence as well as the doubtful findings in such instances make them practically negligible, and certainly no right-minded urologist would consider such meager positive findings sufficient warrant for a nephrectomy. Histologically he finds only round-celled and leukocytic infiltration with fibrosis, but no tubercles. He admits it is a tissue reaction to tubercle bacilli, and is therefore a tuberculous process. Perhaps such patients possess an unusually high degree of immunity which accounts for the benignity of the condition.

In my interpretation of "never," I should have said "rare" instead of "exceedingly rare." The exceptions occur in less than one per cent of cases.

In conclusion, I wish to thank the three distinguished urologists who have taken time off to prepare considered discussions.

BACTERIOPHAGE METHOD OF TREATMENT OF INFECTED WOUNDS*

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THE infected wound is undoubtedly one of the most difficult and most common problems in the history of medicine. The search for the ideal wound treatment is centuries old, ranging all the way from the ashes and natural balsams of primitive man, the incinerated toads and boiling oil of the Middle Ages, to the familiar Bipp treatments and Carrel-Dakin irrigations, employed so extensively in the late war, and the present-day use of maggots. It seems scarcely credible, in our modern stage of scientific advancement, that a method of wound treatment which may revolutionize all our previous concepts could be devised, yet I believe the bacteriophage to be just such a method.

FACTORS IN SUCCESS OF RECONSTRUCTION SURGERY

One of the greatest handicaps of the reconstruction surgeon is the recrudescence of latent infection in such cases. In fact, it can be safely said that the success of reconstruction surgery depends, in many instances, on the method of treatment of the original wound. One of the most frequent forms of bone infection which the surgeon of today may encounter is osteomyelitis. Nearly everyone is familiar with the treatment of this condition proposed by Dr. H. Winnett Orr in 1923. Violating the traditions of free drainage, he packed the saucerized wound with vaselin and vaselin gauze and enclosed it in a plaster cast which was left undisturbed for weeks. Obviously the process was sound, for soon after operation the patient's temperature dropped to normal, and upon removal of the cast the wound was found to be covered with healthy red granulation tissue. However, Orr's explanation (rest, immobilization, and avoidance of reinfection) did not seem to me to account for the marked success of the treatment. After close observation of several cases, I became convinced that some unusual phenomenon was taking place. It seemed to me that practically the same conditions were created under this cast as occurred naturally in the bacterial culture tubes of D'Herelle, the French bacteriologist at Yale, who, in 1921, discovered an ultramicroscopical parasite which appeared spontaneously in a culture of dysentery bacilli and which he called the "bacteriophage." He further demonstrated by laboratory experiment that there were several varieties or "races" of 'phage, each with a preference as to the type of bacteria it would destroy. Was it not quite possible that a native bacteriophage had multiplied and become active under the long-continued dressing of vaselin and vaselin gauze?

By careful laboratory search and tests,† it has been established that a specific 'phage appears spontaneously in about 94 per cent of cases of acute and chronic osteomyelitis. In three of the remaining six per cent in which the 'phage does not appear spontaneously, the laboratory has been able to supply us with a 'phage specific for the organism in question. But in the other three per cent, it has, so far, been unable to do so. This is especially true of the *Streptococcus hemolyticus,* and in these cases we have adopted a policy of watchful waiting. In several instances the desired 'phage has later appeared in the wound spontaneously and healing has occurred. It is hoped that with the perfection of laboratory methods and increased knowledge of the 'phage it may be possible to isolate races of 'phage specific for each bacterium in all cases.

'PHAGE THERAPY IN OSTEOMYELITIS

In cases of osteomyelitis, both acute and chronic, I now make use of the following method of treatment. I do not use iodin or alcohol lest they interfere either with the development of the spontaneous 'phage or with the specific laboratory-bred 'phage after its introduction. In place of the vaselin and vaselin gauze dressing, I am using paraffin with a dash of yellow vaselin, or sufficient vaselin to produce a semi-solid wound tampon. This varies from 90 per cent paraffin—10 per cent vaselin to 75 per cent paraffin—25 per cent vaselin.

The treatment of a group of cases comprising the various complications of osteomyelitis such as compound infected fractures with osteomyelitis, suppurative joints with osteomyelitis, etc., entails a multitude of considerations. In most of these cases we have deep wounds extending into the bone, with varying degrees of infection. The wound dressing, or tampon, should have a degree of solidity sufficient to restrict the tendency of the orifice at the dermis to close earlier than the depths of the wound. Furthermore, this tampon

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